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STEVENS-JOHNSON SYNDROME WITH PULMONARY INVOLVEMENT

GEORGE C. MCELFATRICK, M.D.*
Wilmington, Del.

An opportunity to observe one of the rarer syndromes of clinical medicine arises in the experience of most physicians. We feel that their recognition can be more readily possible if those who see them present them to the attention of the profession.

Stevens-Johnson syndrome, or eruptive fever with stomatitis and opthalmia, is a rare and striking entity. It is an acute condition manifested by headache, malaise, and lesions of the mucous membranes of the eyes, mouth, and urethral meatus. Duration of this condition is several days to several weeks. The exact etiology is unknown, but it is generally agreed that it should be classified with erythema multiforme.

The onset is abrupt with a temperature of 102 to 104 degrees F., headache, chill, malaise, sore mouth and throat. Vesicles then appear on the lips, buccal mucosa, tongue, and pharynx, which later become pseudo-membraneous with or without ulceration. Along with the oral and constitutional symptoms there is a conjunctivitis, rhinitis, and balanitis. In the majority of cases there are also skin lesions typical of erythema multiforme.[1]

In the cases reported in the literature only a few have stressed the association of pneumonitis with Stevens-Johnson syndrome, and the percentage of fatalities that occur despite chemotherapy. Staynon and Warner reported a series of 17 cases with pneumonitis, 14 of which were non-bacterial with an incidence of two fatalities.[2] The Commission on Acute Respiratory Diseases reports 6 cases with pulmonary lesions of non-bacterial pneumonitis.[3] Olsen et al. report a case with pneumonitis which was benefited by streptomycin.[4] Finland et al. report 4 cases with 3 fatalities.[5]

The pneumonitis of these cases is quite similar to primary atypical pneumonia in that prodromal symptoms, absence of physical signs of consolidation, x-ray features, minimal degree of respiratory distress, and absence of bloody sputum, are present in both conditions. Bacteriologic studies in cases of pneumonitis with Stevens-Johnson syndrome have failed to reveal any predominant organism, which is in support of the suspected viral etiology of this condition.

In the fatal cases it is felt that death does not result from the pneumonitis itself, but is due to obstruction of the respiratory passages, with subsequent suffocation. In cases of exfoliative dermatitis there is also desquamation of the epithelium of the trachea, bronchi, bronchioles, and alveoli. From this, it is conceivable that in Stevens-Johnson syndrome the mucosa of the tracheo-bronchial tree is involved in a vesiculo-bullous eruption identical with that seen in the mouth, and that desquamation and obstruction may occur. The degree of pulmonary involvement is apparently independent of the extent and severity of the accompanying pathology. Therapy in all cases seems to be entirely symptomatic.

Following is a case report with definite pneumonitis and survival.

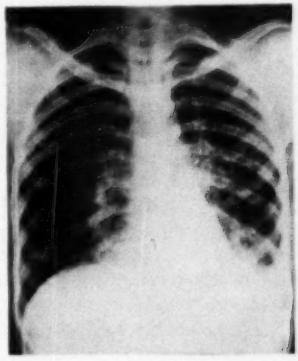

Fig. 1

X-ray of chest reveals pneumonitis similar to a typical pneumonia.

From the Medical Service, Fort Lawton Station Hospital, Seattle, Washington.
*Formerly Captain, M. C., A.U.S., now Resident in Surgery, Delaware Hospital.

CASE REPORT

M.R., a 19 year old white male, was admitted on 6 April 1949 with complaint of cough and pain in the chest. Symptoms started on 2 April 1949 with rhinitis, slight cough, and the usual signs of a beginning coryza. These symptoms became more severe and cough became productive of moderate amount of blood-streaked sputum on 4 April 1949. Sharp substernal pain was present for the 24 hours prior to admission. He had not been able to retract foreskin of the penis for 3 days. Past history reveals that one month prior to admission patient had been in hospital for 4 days with diagnosis of Vincent's stomatitis.

Physical examination revealed a well developed, well nourished white male lying quietly in bed in no acute distress. Temp. 101.6; P-120; R-20; lungs clear. Penis shows moderate amount of swelling of foreskin, no redness, with moderate amount of thick yellow pus exuding from around the glans. Foreskin could not be retracted to see if there were any penile lesions. Examination otherwise negative.

X-ray of 6 April 1949 showed an area of increased density in the left upper lobe 2cm in greatest diameter, with associated hilar drainage. Impression: pneumonitis, cause unknown. Rbc 4,420,000; Wbc 11,600; Hb 13.5; Differential: PMN 74; lymphocytes 20; monocytes 6. Urine: 1.030; clear; acid; albumin-trace; sugar negative; microscopic loaded with Wbc. Serology negative.

Due to continued fever and finding of pneumonitis, patient was started on penicillin 300,000 units every 3 hours on 7 April 1949 at 0900 hours. In the afternoon of 7 April 1949 patient's lips were noted to be swollen and slightly cracked, and small discrete white lesions were seen on the inside of the lips. Patient complained of some difficulty in breathing.

On 8 April it was noted that lesions were spreading throughout the mouth on buccal mucosa and pharynx, a large yellowish white plaque developed on the roof of the mouth, and there was a foul odor to the breath. Routine culture was taken of the mouth lesions which showed no predominant organisms to be present. Patient had a cough productive of moderate amounts of yellowish, tenacious sputum. Patient appeared quite toxic and lethargic. At this time it was also noted patient was developing a severe conjunctivitis of both eyes.

Civilian consultant saw patient on 9 April and concurred with diagnosis of Stevens-Johnson syndrome. It was noticed for the first time that patient had 3 small skin lesions; a 2 x 3cm. macular rash on the back opposite the 2nd lumbar vertebra, on the inner side of the left ankle, and another just above the left knee. Diphtheria culture was taken from the throat, which later proved to be negative. Patient received borie acid soaks for conjunctivitis, and normal saline mouth wash. Patient was having some difficulty in respiration, but no cyanosis was evident.

Patient showed some improvement on 10 April although there was no change in the lesions of the mucous membrane or the skin.

On 11 April it was noticed that the lesions in the mouth were not as discrete, but there was a generalized grayness of the mucous membranes of the mouth. Sputum did not appear to be as thick or tenacious. Patient had been receiving intravenous fluids since 8 April and was not able to take fluids by mouth until 13 April. Patient was started on sulfadiazine gm. 1 every 4 hours on 13 April. Penicillin was discontinued 14 April as patient still had elevation of temperature to 101 degrees. The lesions of the mouth and skin, the conjunctivitis, and balanitis all continued to show improvement, and all lesions were completely healed by 25 April. Sulfadiazine was stopped on 16 April and patient's temperature returned to normal on 18 April and remained normal thereafter.

SUMMARY CONCLUSION

A case of Stevens-Johnson syndrome with associated pneumonitis is presented. This is an additional report of association of the two conditions in the hope that lung involvement will be watched for in these cases, and therapy instituted in the hope that a therapeutic agent for this condition may be discovered.

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MIKULICZ'S SYNDROME

Report of Two Cases

GEORGE B. HECKLER, M.D.*
Wilmington, Del.

In 1888 von Mikulicz described a symptom complex which was to become known as Mikulicz's disease. The disease entity which he suggested is now considered as consisting of lacrimal and salivary gland enlargement, which in most instances is painless. The histological appearance is that of intense round cell infiltration with varying amounts of glandular atrophy and interstitial fibrosis, and without epitheloid tubercles or giant cells. The enlargement may effect one, several, or all of the salivary glands and/or the lacrimal glands. The involvement may be of all gradations. Mikulicz's disease proper is considered to be benign, self-limiting, and of unknown etiology.

Since Mikulicz described his original case, many cases have been reported in the literature. In most instances one of several possible etiologic causes has been found responsible for the symptom complex. Some of the underlying causes have been progressive in nature, and a few cases have had a fatal termination. This latter group, in which an etiologic factor can be demonstrated, is correctly labelled *Mikulicz's syndrome*. The histological picture in this group is essentially the same, but giant cells and epitheloid tubercles are frequently present, and pathology can be frequently found in other organs. The syndrome, in contradistinction to the disease, is not rare in occurrence. Many of the cases originally called Mikulicz's disease on re-evaluation have fallen into the Mikulicz's syndrome group.

In 1927 Schaffer and Jacobsen[1] presented a classification of Mikulicz's disease which seems to be all-inclusive. These authors stress that the classification of Mikulicz's disease proper should be reserved for those cases in which no underlying etiologic agent or associated disease is found.

Classification of Mikulicz's disease by Schaffer and Jacobsen:

A. Mikulicz's disease
   1. Familial
   2. Mikulicz's disease proper

B. Mikulicz's syndrome
   1. Leukemia
   2. Tuberculosis
   3. Syphilis
   4. Lymphosarcoma
   5. Sarcoidosis (febris muco-parotidea subchronica)
   6. Toxic reactions
      a. Iodides
      b. Lead
      c. Arsenic
   7. Gout

Most authors agree that this classification is not entirely satisfactory. Jackson[2], Heaton and Shannon[3] have indicated three major faults: (1) the cases which have been diagnosed as familial Mikulicz's disease have been simple hypertrophy of the salivary glands. Simple hypertrophy of the glands may be not infrequent in the colored race. (2) Lead and arsenic have not been shown to be responsible factors in the production of Mikulicz's syndrome. (3) gout involving the temporo-mandibular joint has been incorrectly confused with Mikulicz's disease and/or syndrome.

The preceding classification readily indicates that the disease may be localized or a part of a generalized process; therefore, the therapy and success of therapy varies widely. Miller, Eusterman, Leddy[4] and other investigators have recorded good results with x-ray therapy; Jackson has reported a case of Mikulicz's disease proper successfully treated with penicillin. Local excision of the glands has been carried out; antibiotic therapy and iodides have yielded a good response. In the following two cases of Mikulicz syndrome penicillin therapy in Case No. 1 was followed by a remarkable response within a few days; and local excision of a lacrimal gland in Case No. 2 has not been followed by a recurrence at the date of writing, four months from the time of excision.

*Resident in Medicine, Delaware Hospital.

CASE 1

A 34 year old colored female (A.S. #165531) was first seen in the Clinic with the complaint of blurred vision of the left eye and painful swelling of the left parotid gland of two weeks duration. She returned to the Clinic one week after the initial visit, and because her symptoms and the objective findings had markedly increased, she was admitted to the hospital on September 29th. There was a history of trauma to the left temporal area one year prior to the onset of the present episode. This was not considered to be a contributing factor to the present difficulty.

The abnormal findings on physical examination were limited to the head. There was total loss of vision in the left eye, and vision in the right eye was impaired. The left eye showed a marked degree of proptosis. The eye was fixed in a downward and lateral position. The eyelid could not be closed over the eye, and when the upper eyelid was retracted the edge of a large lacrimal gland was evident. Examination of the left eye ground revealed marked venous distention but was otherwise normal. There was slight exophthalmus of the right eye with marked puffiness of the upper eyelid. An enlarged lacrimal gland readily protruded when the eyelid was retracted. The right eye ground was normal. Both parotid glands were abnormally large; the left parotid gland was very tender to touch and the enlargement extended beyond the zygoma and below the angle of the mandible. The mouth could be opened approximately three-fourths of an inch with difficulty.

The patient was afebrile. The pulse rate and blood pressure were normal. Laboratory studies revealed: RBC 3,900,000; Hb. 76% or 11.9 gm., WBC 4,400; Polys. 59%; Lymphs. 31%; Mono. 4%. Sedimentation rate 26.0 mm. in one hour. Fasting blood sugar 90 mgm.%. The urinalysis was essentially normal. Skull and chest x-rays were within normal limits. Serology: Mazzini, positive; Kolmer-Wassermann 3 plus; Kahn standard, positive. The spinal fluid was clear, and under normal pressure. Other studies on the spinal fluid showed a two plus Pandy reaction, Wassermann 4 plus, and a colloidal gold curve 123321000.

In the presence of the symptom complex and the positive serology, it was felt that this clinically represented a Mikulicz's syndrome. A course of antiluetic therapy, with penicillin as the agent, was instituted. On the fifth day of therapy a definite response was quite evident. Vision in both eyes had returned to normal; the left eye had markedly receded from its abnormal position; extra-ocular motion was normal, and the eyelid closed with ease. The swelling in both parotid areas was considerably diminished and the left parotid was no longer tender. The patient was discharged on the tenth hospital day, and at that time there was no abnormal swelling of either parotid gland and the mouth could be opened without difficulty. Eye motion, position, and vision were normal, and the lacrimal glands were relatively normal in size.

This patient was subsequently followed in the Clinic, and had no further difficulty with vision or swelling of the salivary glands.

CASE 2

The patient, (E.W. #168152) was a 24 year old colored female. During a routine physical examination in the Clinic in May 1947, it was noted that she had puffiness of both eyelids. She was symptom free at that time. In August, 1949 attention was again drawn to the drooping and protrusion of the eyelids. She had noted excessive lacrimation from the left eye for two months preceding this examination. It was also noted that there was enlargement of both parotid glands and the right submaxillary gland. She did not complain of pain or tenderness in these glands.

The past history was significant in that she was diagnosed as having syphilis in 1946. Subsequent to that diagnosis she received 56 weekly injections of mapharsen and 30 injections of bismuth subsalicylate. In 1948 she also received a 10 day course of penicillin therapy. In 1946 a routine chest x-ray showed a fibroid type acid fast lesion in the right apex of the lung field. This lesion has remained unchanged since that time. Sputa and gastric washings have been negative for acid fast bacilli. PPD skin tests, first and second strengths have been negative.

General physical examination was normal. There was no generalized lymphadenopathy.

The spleen and liver were not palpable. Examination of the eyes revealed normal vision and normal ocular motion. Both upper eyelids protruded, and beneath the left upper eyelid there was a firm, oval-shaped mass which was non-tender. When the eyelid was retracted the mass was noted to be a greatly enlarged lacrimal gland. Similar findings were noted on the right, but enlargement of the right lacrimal gland was not as pronounced. Bilateral parotid gland enlargement was evident as well as right submaxillary gland enlargement, the glands were non-tender. Examination of the eye grounds did not reveal any abnormalities.

In January, 1950 the patient was admitted to the hospital for biopsy of the left lacrimal gland. Examination of the eyes showed that the process had been stationary over the past 5 months.

Laboratory studies: Hb. 75% or 11.9 gm.; WBC 5,000; Polys 59%; Lymph. 34% Mono. 4%; Eos. 2%; Bas. 1%. Total serum protein 8.62 gm., Albumin 5.26 gm., Globulin 3.36 gm. x-rays of the skull and hands were normal. Serology: Mazzini 4 plus; Kolmer-Wassermann, 4 plus. The urinalysis was normal.

The left lacrimal gland was excised. It was found to be extremely enlarged and very firm in consistency. Grossly, the gland was hard and the cut section was a gray-white color. Microscopic study revealed a distortion of the lacrimal gland by increased fibrous tissue, marked round cell infiltration and occasional giant cells with resulting parenchymatous destruction.

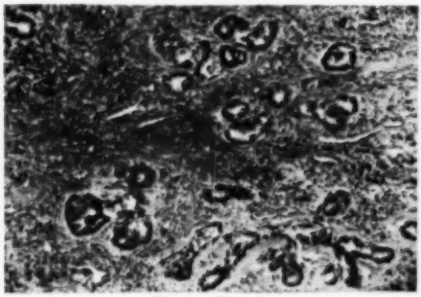

Fig. 1: Case 2

Area in lacrimal gland showing fibrosis, separation of acini, moderate round cell infiltration.

The exact etiologic cause of the glandular enlargement in this case can not be recorded with certainty in view of the presense of possibly two disease processes capable of resulting in a Mikulicz's syndrome. Further follow-up of this case may reveal the actual etiological factor. At present there has been no local recurrence.

SUMMARY

1. Mikulicz's disease is a rare entity, generally considered to be a benign, self limiting disease, producing painless enlargement of the lacrimal and salivary glands with a characteristic histological picture, and is of unknown etiology.

2. Mikulicz's syndrome is more frequent in occurrence and produces similar clinical enlargement of the lacrimal and/or salivary glands, but it is secondary to, or associated with, other disease processes, e.g. leukemia, tuberculosis, syphilis, lymphosarcoma, sarcoidosis, iodides. The syndrome because of its secondary causes, may terminate fatally.

3. Two cases are presented as Mikulicz's syndrome: The first which was secondary to syphilis and responded dramatically to antiluetic therapy. The second, which may be secondary to syphilis or tuberculosis, has shown no local recurrence after surgical excision of one involved gland.

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HYPONATREMIA IN CARDIAC FAILURE
A Case Report

LEWIS B. FLINN, M.D.,*
and
JOHN H. BENGE, M.D.,
Wilmington, Del.

That depletion of body sodium concomitant with an insufficient cardio-circulatory mechanism effects development of tissue edema is recently an acknowledged fact. This state closely simulates the edema of cardiac failure where

*Director, Medical Service, and Resident in Medicine, respectively, Delaware Hospital.

an excess of the same electrolyte plays a primary causative role. The clinical differentation of these diametrically opposed states is not always possible. However the deficient state is more prone to occur in the face of a low sodium diet and with the use of frequent mercurial diuretics. That it may be produced insiduously without vigorous therapy is exemplified by the following case report.

CASE REPORT

The patient, (R. McC. #169385) a white male of 50 years entered the Delaware Hospital for the first time on March 8, 1950, with the chief complaint of a severe "pressure-like" pain located substernally. The patient had been seen by one of us in August 1949 when he gave a very typical history of angina on effort for the preceding three months. An electrocardiogram at rest showed only slight evidence of coronary arterial disease, but after exercise there were definite ST changes suggesting coronary insufficiency.

His general condition improved under rest and customary therapy. Other symptoms suggested biliary tract disease, and a cholecystogram on February 20, 1950 demonstrated gall stones and a poorly functioning gall bladder. His present episode was attended by the physical findings and laboratory evidence of a posterio-septal myocardial infarction. At this point the signs of decompensation were bilateral pulmonary rales and a moderately enlarged liver. There was no pretibial edema nor pleural fluid. Therapy included sedation, oxygen, papaverine, dicumarol, bed rest and a low salt diet (2 to 4 grams daily). Gradually there developed marked pretibial edema and on March 24th he was given 1 c.c. of thiomerin and digitalis begun. This resulted in little diuresis and so on March 27th a 2 c.c. dose was given with a moderate response. The next day there developed a slight dermatitis believed to be due to bed linen. On March 31st a third dose of the mercurial was given resulting in a minimal response. The dermatitis increased and it was deemed best to discontinue the mercury. Ammonium chloride was substituted on this day in a dosage of 4.0 grams a day. During the next two weeks he improved steadily so that on discharge there was no pulmonary edema, moderate pretibial edema, a slightly enlarged liver and no pleural fluid. The dermatitis was clearing.

His urinary output was normal and a slight azotemia noted early had been reversed. As evidenced by the ability to concentrate urine, the renal status appeared good. There was a trace of albumin on one occasion. His blood pressure had become stabilized at 90/60.

While at home during the next 8 days he was at bed rest on a low salt diet, and was taking digitalis and ammonium chloride. After four days of this regime he noticed increasing dyspnea and ankle edema. He was readmitted on April 20th, 1950 in marked respiratory distress, cyanotic, with marked peripheral edema and with bilateral pleural fluid collections of minimal amounts. The hepatomegaly had increased but there were but few pulmonary rales.

In the next few days his physical status improved slightly under oxygen and with strict bed rest. His edema however progressed slowly to involve the sacral and scrotal areas and then the thorax. By his 8th hospital day he began slowly to lose ground, becoming progressively more disoriented, developing a severe cough and eventually Cheyne-Stokes respirations. On the 14th hospital day the edema had become so massive and his urinary output so low that it was decided to attempt diuresis with hypertonic glucose solution, which was given on that day. 300 c.c. of a 25% glucose solution was given intravenously and followed with 2000 c.c. of a 10% glucose solution. The patient progressed to coma under this therapy and urinary output decreased further. It was decided that further attempts to reinstate urinary flow with glucose were futile and the syndrome of salt depletion considered, although the serum sodium was only slightly below normal (129 mille-equivalents per liter). Therefore 300 c.c. of 5% sodium chloride was given intravenously. Within a few hours the patient began to arouse himself from coma and it was noted that the urinary output was increasing. In spite of the high intake of sodium chloride, the next day his chlorides and serum sodium were markedly depressed (sodium 114 mille-equivalents, chloride 460 mgm.%). Similar therapy was

carried out for the next three days with the addition of a mercurial diuretic intravenously (salyrgan).

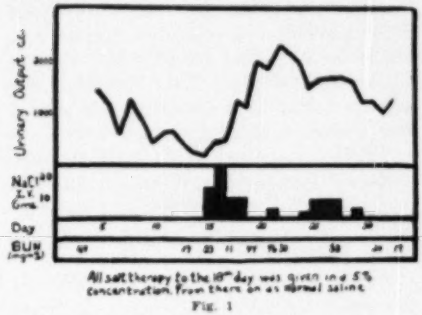

FIG. 1

His clinical and laboratory response were very satisfactory under this regime. Especially noted were the decrease in edema and increase in urinary output. His response continued for three days following cessation of therapy with salt. At this time he developed a severe monilid infection of his oral and pharyngeal cavities and refused to take oral nourishment. Electrolytes and fluids were given intravenously as seemed indicated from clinical studies from day to day. Although his urinary output remained well above normal he began to deteriorate and died on the 39th hospital day. It is interesting to note that his pulmonary fields remained remarkably free of rales until the last few days of life and stood out in sharp contrast to the other signs of edema. Pericardial friction rubs had appeared on several occasions.

Post mortem examination revealed an old posterior-septal infarction with two early septal aneurysms and multiple fresh pulmonary infarctions. The coronary vessels were each occluded by organizing thrombotic material. There were mural thrombi in the left ventricle and right auricle. The renal tubules showed a diffuse intracellular change characteristic of that seen following hypertonic intravenous therapy.[1,2]

DISCUSSION

Since the days of Hope, the concept of the mechanism of cardiac edema has been constantly modified to explain clinical and experimental observations. Krogh and Landis cleverly explored the role of increased venous pressure or the so-called "backward failure" theory. More recently workers have demonstrated that sodium retention due to decreased glomerular filtration which is in turn due to decreased cardiac output played a prominent part. Their work has provided the so-called "forward failure" theory. Later with the advent of modern methods of treatment a third factor, namely sodium depletion, has been shown to be capable of initiating collection of tissue fluids. Still to be investigated are the contributions of the hormonal control and physiologic state of the renal tubular epithelium.

Fishberg[3] has recently pointed out that an increasing number of cardiacs under modern therapy enter a deleterious phase associated with an impairment of renal function previously considered adequate. His cases exhibit pathologic renal tubular changes which he calls "regressive." He indicates that the cause of such changes has not been explained. Some of these patients respond to less intensive mercurial therapy and the administration of sodium chloride. In this case we found similar renal tubular damage, but feel it cannot be due to mercurials. Whether it represents changes due to hyponatremia or hypertonic fluids is not known.

The patient's BUN rose during salt therapy and immediately following diuresis but with continued fluid loss became normal. This has been reported previously. Again the cause for this is not evident. Urea has been used as a diuretic with the explanation that in concentrations exceeding maximal tubular reabsorption it "drags" water and electrolytes with it. Apparently the converse is not true.

The most interesting feature of this case was the insidious onset of hyponatremia in the absence of either marked sodium depletion from the diet or prolonged mercurial diuretic administration. A possible etiologic agent was ammonium chloride.

SUMMARY

A case of hyponatremia developing in a cardiac patient has been presented, with the therapy and clinical course.

The factors of the production of cardiac edema have been mentioned.

It has been emphasized how unsuspectingly

a state of hyponatremia may develop.

An unusual feature, namely, proximal tubular epithelial damage characteristic of hypertonic glucose, was associated with this case.

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CLINICOPATHOLOGIC CASE RECORD

WILLIAM J. HOOPES, M.D.*
and
C. T. CHOW, M.D.
Wilmington, Del.

Presentation of Case**

DR. HOOPES:

This sixty year old negro man—a delivery man and shipping clerk—with the chief complaint of epigastric pain had had intermittent pain for the six weeks previous to his admission, pain spreading up and down beneath his sternum. He had the pain every day and was unable to continue work because of it. The pain was relieved for a short time by eating food but always recurred quickly.

In the two or three days prior to admission the pain was increased and a temperature elevation was noted by the local medical doctor. There had been anorexia and twenty pounds weight loss in the past month.

The past history and system review showed only some exertional dyspnea, precordial pain and palpitation during the past few weeks. No cough or edema. No nausea or vomiting. Some bright red blood in stool lately.

Physical examination revealed pulse 120, irregular. Blood pressure 85/50. Respirations 30 and temperature 100.8°F. There was opacity of the right lens. Transient rales over the right lower lobe laterally which cleared on continuous deep respiration. Heart—apical rate 160, radial pulse 120 and irregular. Systolic murmur in the mitral area. The abdomen showed marked epigastric tenderness with muscle guarding, no spasm. Most comfortable while sitting up leaning forward. Rectal examination showed the prostrate to be firm, enlarged and non-tender. Some tenderness at finger tip on upward and forward pressure.

Laboratory data: Red blood count 3.0 million; hemoglobin 9.1 gm. (58%) polys. seg. 85%; polys non-seg. 4%; lymphocytes 8%; monocytes 3%; sugar 112 BUN 27.2 mgm.%; Wassermann 4 plus; Kahn positive. The urine is turbid, dark amber; specific gravity 1.024; reaction acid; albumin plus 2; sugar and diacetic negative; 1—3 white blood cells; numerous hyaline casts; occasional epithelial cells and a few mucus threads. Febrile agglutination was negative except for Proteus OX19, 1:40.

X-ray revealed cloudiness over both diaphragms. The costophrenic angles obliterated. Both hilar shadows increased with accentuation of vascular markings fanning out from both hilar zones, especially toward bases. Trachea and mediastinum are in the mid-line. The heart shadow shows marked general enlargement. Heart measurements: oblique 18; right border 7; left border 11.1; right auricle 6.5; left ventricle 6.2; arch 7 cm. Cardiothoracic ratio 18.5 to 20. Diagnosis: general cardiac enlargement with pleural effusion of both bases and some passive congestion.

The electrocardiogram was interpreted as low voltage which is abnormal in four out of five cases and which is definitely classed as abnormal due to elevation of ST seg. in I and II. This elevation does not somehow suggest pericarditis, but it is not normal and a suspicion of that condition should be entertained. It is definitely an abnormal tracing.

There was a persistent elevation of temperature during the hospital course, often to 102° F. or above. Gradually temperature fell in two days prior to death. Patient ran a rapidly downhill course with frequent periods of disorientation. One day before death a pericardial friction rub was heard. That day he became very dyspneic and was placed on oxygen. He expired on the 5th hospital day.

DIFFERENTIAL DIAGNOSIS

DR. CHOW:

In this case there are three major sites of involvement. As I see it, the problem is to determine the type of involvement in each site, if possible, and then to determine whether any or all are related. The three major sites

*Internes, Delaware Hospital.
**Delaware Hospital Case No. 64216 presented at Staff Clinical Pathological Conference.

to my mind are heart, gastro-intestinal tract, and lungs.

Let us analyze the symptoms and signs. Consider the heart first. The patient's illness started with precordial pain, palpitation, and dyspnea. In this age group the most probable explanation for such symptoms would be myocardial infarction. But it seems unlikely that this patient had myocardial infarction. Chest x-ray shows that there is pleural effusion at both bases. Pleural effusion is rarely present in myocardial infarction, and the heart is dilated to such a degree that it exceeds that usually seen in myocardial infarction unless it is associated with pericarditis. A persistent elevation of temperature to 102° and a pericardial friction rub are not present in most cases of myocardial infarction. The EKG is not suggestive of myocardial infarction. Did he have hypertensive or arteriosclerotic heart disease?

We do not know whether he had had hypertension before. If he had had it in the past of a sufficient degree and duration to result in a heart of such size, surely he would have been in severe congestive heart failure when the blood pressure dropped to 85/50. He did have dyspnea but no peripheral edema, and liver enlargement was not noted. Only transient rales were heard in the right lower lobe and cyanosis was not mentioned. In a patient with a positive Wassermann at that age, syphilitic heart disease is a possibility. We do not know whether he had been or was being treated with penicillin or arsenicals. A probability is pericarditis which could account for the sequence of most of the events recorded, notably the prolonged shifting substernal pain and the low blood pressure which could well be due to fluid in the distended pericardium impeding the entrance of blood from the great veins, thus lowering cardiac output. The increased heart rate, to 160, might be explained as compensatory for a diminished stroke volume. The pulse deficit and also the friction rub could be explained by pericarditis. The EKG pattern, furthermore, is somewhat suggestive of that condition.

To be discussed is the respiratory tract. The shifting substernal pain may have been due to pleurisy and the chest x-ray shows evidence of some pleurisy. The pain may have been due to the invasion of nerve trunks in the mediastinum or chest wall. While it is true that pleurisy very frequently jumps from one place to another, I cannot explain the shifting nature of the pain on that basis. Furthermore, the pain was not likely due to the accumulation of fluid in the chest. The x-ray shows that both hilar shadows are increased with accentuation of vascular markings. Could it have been acute passive congestion in mild form, because of an increase in pulmonary blood volume?

Pericarditis is probably never a primary disease. The six most frequent pathologic conditions with which it is associated are: (1) rheumatic fever, (2) post-pneumonic empyema, (3) pulmonary tuberculosis, (4) chronic nephritis with uremia, (5) coronary thrombosis with myocardial infarction, (6) perforating wound of the thorax. I think that with rectal bleeding however, a lesion in the G. I. tract is a better possibility.

The third major site I am going to discuss, therefore, is the gastro-intestinal tract. The bleeding could have come from hemorrhoids but it evidently occurred simultaneously with the cardiac symptoms, so I think there might be some relationship to them. At any rate I do not think the blood was coming from hemorrhoids. Could this have been peptic ulcer which is the common cause of massive hemorrhage, accounting for probably 60 to 70% of bleeding in the upper G. I. tract? I suppose that a simple peptic ulcer cannot be ruled out but such a lesion seldom perforates the pericardium; so it could hardly explain a pericarditis as well as gastro-intestinal hemorrhage. It might have been a thoracic stomach although that is very rare. The location of the esophagus posterior to the pericardium must be borne in mind. There the esophagus is normally adherent to the posterior aspect of the pericardium, it is also in contact with the pleura just before it passes thru the diaphragm. Could this have been a herniated short esophagus with an associated esophagitis, ulceration, and fibrotic change? Most

cases of esophageal ulcer occur in the lower three inches of the esophagus, usually within an inch and a half of the cardia, where a perforation may be peritoneal, pleural, pericardial, or mediastinal.

The symptoms associated with abdominal type of perforation do not differ materially from those of perforation of gastric ulcer. Acute perforation above the diaphragm may produce a clinical picture suggestive of a pulmonary or cardiac catastrophe with acute severe chest pain and shock. I do not favor this diagnosis because of the insidiousness of the patient's symptoms. Could this have been a tertiery syphilis of the esophagus considering the four plus Wassermann? Such a lesion would obviously be a very remote possibility even in a known syphilitic. A more likely diagnosis would be cancer of the G. I. tract and there is some evidence that this patient had cancer. In the first place a patient in the old age group with a short history of gastric distress and recent weight loss is more likely to have a malignant condition than benign. On this basis I am slightly inclined toward cancer in the upper G. I. tract. It is difficult to make a differential diagnosis between a primary cancer of the esophagus with extension into the stomach or vice versa. Because of the lack of serosal covering of the esophagus, cancer in the esophagus is liable to spread by local infiltration. But the patient had no dysphagia, the most characteristic symptom of cancer of the esophagus. This might be explained by a cancer which did not grow intra-lumenal but did extend outside of the esophagus to the pericardium thus causing a pericarditis.

Dr. Chow's Diagnosis: (1) Carcinoma of the esophagus with extension to the pericardium causing pericarditis. (2) Syphilitic esophagitis with ulceration into the pericardium. (3) Cancer of the stomach with extension to the esophagus and perforation of the pericardium.

PATHOLOGICAL DIAGNOSIS

Epidermoid carcinoma of esophagus with extension to pericardium. Acute purulent pericarditis.

AN OPHTHALMOLOGY PROJECT IN ALASKA*

DAVIS G. DURHAM, M.D.**
Wilmington, Del.

This summer I was fortunate enough to be included in a project in eye research problems in Alaska. I thought that I would describe briefly the project and then show you some pictures of the eye conditions we found there, and also some of the local scenery.

The problem that we were particularly interested in was phlyctenulosis, which simply means a small blister or wart on the eye. This is usually on the conjunctiva, on the cornea, or both.

Unfortunately, in the past there never has been any work done with the Eskimos. In fact, there has never been any qualified eye man to go with the necessary equipment into the region to examine them.

The project came about through a friend of mine who was associated with me in the Army, who is now practicing in Anchorage. He had done the groundwork for the project in that he had visited practically all the Eskimo villages throughout Alaska and set up the organization for this particular project.

We were located in a little village of Sitka, which was originally the Russian capital of Alaska and is on the southeastern part of the coast of Alaska on a small island. It was formerly a naval base during the war, and after the war it was abandoned. It was taken over as an Alaskan native school.

We had approximately 350 Eskimo children there, and therefore we were able to examine all of them in a month's time, and do that much more efficiently, without transporting our equipment and so forth to all the villages.

We had associated with us four eye doctors, and a bacteriologist. We drew on the services of a tuberculosis specialist, and, in addition, the dietitian who was associated with the school. The sponsors of the project were the Alaskan Native Service and the Department of Health of Alaska.

The equipment that we required was mainly bacteriological and that necessary for the eye

*Read before the Medical Society of Delaware, Wilmington, October 12, 1949.
**Assistant in Ophthalmology, Delaware Hospital.

examination, which included particularly a slit lamp bio-microscope; also all the necessary microscopes and Petri dishes and culture media and so forth, which are quite a problem when you have to start more or less from scratch.

Also, we had special cameras to take eye pathology, and, in addition, the school furnished us with a house, and a jeep for transportation. So we were very comfortable and had an ideal location for such a project.

The etiology we were looking for, which was really the basis of the project, before we could do any treatment, was quite a formidable one. Our current thought was that it was due to tuberculosis, not a specific infection of the eye with the t.b. organism, but rather an allergy to the tubercular protein. Other possible etiologies were diet, which we more or less ruled out on the basis of the fact that most of these children had been living in the school for up to three years and had been getting an adequate diet, although, like most children, they had a great deal of candy and carbohydrates; still, we did not feel that the diet was a contributory factor to the condition.

Also, vitamin deficiencies were considered, but we could find no evidence of this etiology.

The serology was checked on every child, and without exception the Wassermann reactions were all negative. Also, we had chest x-rays on every child, and without exception there was some evidence of tubercular infection in every child. That was either in the lungs, with a healed primary lesion or an active lesion, or bone tuberculosis, or tuberculosis of the glands.

Also, we checked the tuberculin sensitivity on every child, and again without exception every one of them was positive to the first strength tuberculin, with the exception of two out of the 350 who were positive to the second strength tuberculin.

(Slide) This first slide is a map of Alaska, which shows the location of the homes of all the children, so that you can see they were well spotted from all over Alaska, from the Eleutian Peninsula all the way up to Nome and Point Barrow. Point Barrow is all the way at the top of the map and doesn't show. We were located in Sitko, the southeastern portion right about where the dot is, which was the location of the school.

This is Nome here, and Fairbanks is located here, and Anchorage is right in this region here. This is the Aleutian Peninsula.

(Slide) This shows the group that we had who made up the project. This is Dr. Fritz, who organized the project. He is now practicing in Anchorage, Alaska. I am next to him. This is Dr. Thygeron, who is another doctor with whom I was associated in the Army, who is recognized today as the world's authority on bacteriology of the eye. So he was the main sparkplug of the organization.

This doctor (indicating), you might be interested in knowing, is a resident from Duke University in eye, ear, nose and throat. This is a new experiment which is being tried out in which the residents in that service in Duke University are rotating to Alaska, spending six months there. The training which they get is really marvelous. The amount of pathology, the amount of surgery which they are able to do far surpasses anything they would be able to do at Duke in the same period of time. They get one year of training at Duke and then they get six months in Alaska, and then go back to Duke for the finishing of their service, which is another year and a half, I believe. They get all their travel expenses paid, and, in addition, they get $200 a month, which is a pretty nice salary.

The three girls were students in the school. They were all about 18, and they were of great help to us with the names and handling the children.

(Slide) This is a typical picture of one of the conditions, phlyctenulosis, in which we were particularly interested. This girl had an acute lesion in her right eye, and you may be able to just barely see the little scarring in the cornea which is the end result of previous attacks. In other words, they get an attack and it will last several months, and then it will clear up, and then it will have a tendency to recur. She had an active tuberculosis in the glands of the neck and showed positive evidence of tuberculosis in the lungs.

(Slide) This is a closeup of the same condition which shows the bulbar conjunctiva in-

jection with the limbal vessels coming in, and in this area you may be able to recognize a grey haze, which is the scarring resulting from previous attacks of the phlyctenulosis.

(Slide) This slide shows an acute attack of the lesion, and there are two leashes of vessels coming in in this area together with some scarring from a previous attack. This particular attack was less than 12 hours old and occurred while we had been checking the girl.

(Slide) This girl is in her early 20's and is completely blind for industrial purposes. This is the unfortunate end result of the condition, for many Eskimos are industrially blind by the time they reach adulthood. In the past this has been blamed on snow blindness, but we now know that this is not true and that it is due to this condition of phlyctenulosis. This girl has less than 20/400 vision in each eye, and I think you may possibly be able to see the grey haze that is present in the cornea. She has no acute attack at the present time. The eyes are quiet, and the vascularity of the cornea is not present and therefore she will be a good candidate for a corneal transplant. When we left, this was being arranged to be done some time in the future.

(Slide) This is a close up of one of her eyes, which may show just a little better the grey haze that is present in the cornea, which is a factor that reduces the visual acuity.

(Slide) This again is another young girl. This shows a quiet eye with the corneal scarring and again an active eye on the left side. The vessels you see coming in, grow into the deeper layers of the cornea. Whenever the cornea becomes vascularized, it leaves scarring and the end result is reduced visual acuity.

(Slide) This is a picture of an acute attack, and I think you can see from the light reflection the unevenness of the cornea due to the vascularity. A normal cornea will show an even light reflection, which this eye does not do.

(Slide) This is another acute attack which has not progressed too far into the cornea but does show scarring from previous attacks.

(Slide) In determining the etiology of the condition, we were looking for any possible cause, so that in examining the children we not only examined their eyes, but also the nose, the ears, throats and any possible source of infection that might be contributing to the condition. We took cultures, both blood agar and mannitol, both of the conjunctiva of each eye and of the lids of each eye, together with the nose and any lesions such as this.

Also, as I mentioned before we did tuberculin skin test, chest x-rays, serology, and also staphylococcic sensitivity on each child.

(Slide) This is a picture of the same girl, which shows marked blipharitis. You can see crusting of the lids, and it has not affected her eyes at all. Her eyes are perfectly quiet. She has a lesion in her left nostril, too, and then she had the lesion behind the ear which we just showed. We did not feel that these lesions had played any part in contributing to the phlyctenulosis.

Also, of course, we examined the under portion of the upper lids of every eye, and just by accident we examined this child and found the typical picture of vernal catarrh, an allergy that we see very prevalent in this country but very rarely in Alaska. This was an asymptomatic condition, but it just shows the typical cobblestone appearance of the conjunctiva which results in intense itching.

(Slide) This is a tuberculin reaction on one of the patients which we did, which shows the rather marked positive reaction. We checked every one of the children for tuberculin sensitivity, and, as I mentioned before, every one was sensitive to the test.

(Slide) This shows a typical Eskimo child, which I put in to show how they do look. She has no ocular involvement. Out of the 350 children we examined, we found over 100 positive cases with no trouble at all. We were looking for 100 positive cases to study completely, and so I would judge that probably at least 50 per cent of the children did show this infection.

This girl, incidentally, has a nyphosis from a tubercular lesion, which unfortunately, is very, very common in that region with the Eskimos.

There was on this same island, in association with this project, a 150-bed tuberculosis of the bone hospital, and this particular girl had been a patient there.

(Slide) This shows the typical scenery. We were on a little island opposite the village of Sitka, and the scenery is just unsurpassed for beauty. It is typical of Norway in that there are many fjords, and the timber on the mountains comes right down to the water's edge. The mountains are snowcapped, and it is just untouched as far as the natural resources go. You may be able to see a seaplane right here in the foreground, because aviation is the only means of getting from one place to another, unless you are not in a hurry. It takes two days to get to the mainland by boat, but we could get over there in an hour by plane.

(Slide) This shows the fishing fleet at Sitka. Fishing was the main occupation of the natives, and salmon fishing played a great part in the fishing industry.

(Slide) This is one of the fishing boats along towards twilight. They are throwing out their nets to catch some of the fish. They go up to the little fjords, as the salmon run up the stream to go back to spawn to the fresh water streams in which they were born.

(Slide) This shows again a little fishing boat and it is typical of the weather which we had there. It is grey and overcast most of the time. When we did get a nice day, we would take off and enjoy the surrounding country, and then we would work all the other days. Out of the month's time we were there we had approximately three or four really nice days.

(Slide) This is a picture inside the cannery which shows the salmon. The fishermen get 35 cents a pound for the salmon, so you can understand why it costs so much, canned, in this country.

(Slide) This is added just to show the twilight. This was about 10:30 in the evening. One of the hardest things to get used to was going to bed at night and having to pull the blinds down. The first night we didn't do it and woke up about two o'clock in the morning thinking it was time to get up. There is very little night at all, and a little further north they have midnight baseball games on June 21.

(Slide) This shows another view of the airplane, which is the main means of transportation. These are marvelous pilots. They fly these little Grummans and in 17 years they have not had a fatal accident. They fly underneath the overcast, and since the overcast is usually about 500 feet, and, of course, the mountains are very much higher than that, they fly until they find a pass and scoot through. If one pass is covered over, they go to another one, and if they can't get through, they put the plane down on the water and wait until the overcast lifts. They are very, very careful and are very good.

(Slide) This shows the landing ramp at Sitka, and we were on the island in the background. The mountain behind that is a volcano mountain which is rather typical and is called Mount Edgecomb.

(Slide) This shows a fresh water lake up in the mountains which we used to hike up through, and the rainbow trout you get out of there are just terrific. Most of these fresh water lakes have never really been fished to any great extent. You can hire a plane and fly in to a lake for a day, and they will fly all the equipment. You get to waters that are just untouched as far as fishermen go.

(Slide) This shows some of the timber. The timber is green the year round, it is nearly all evergreens, and it grows to very great height.

(Slide) No trip to Alaska is complete without totem poles and these are totem poles right here in the village in which we were.

(Slide) This shows the scene in the park near the totem pole. This was one of the unusual days that we had. Unfortunately we didn't get many sunny days.

(Slide) This shows the picture from the airplane on the way home as we left Alaska. The planes scoot by the mountains, and you feel as though you could reach out and touch them.

There are one or two things that impressed me very much about the project. First of all, that you are able to conduct your practice in a town like Wilmington and then in a matter of 36 hours, you are in a far portion of the world where there are terrific needs for medical attention. Now that we have fast plane transportation, we probably could go to any part of the world and take part in such projects.

The other is the terrific need for such investigation and the need for adequate medical studies and personnel in these backward areas.

Until the doctor who organized the trip, Dr. Fritz, went to Alaska in the last year or so, there was no eye, ear, nose and throat man practicing in Alaska who was qualified to examine and study these conditions or to do any surgery.

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Dover.

Committee on Fractures and Other Traumas
American College of Surgeons
THE CARE OF HAND INJURIES

II

REQUIREMENTS OF EARLY DEFINITIVE TREATMENT

I The first-aid treatment of hand injuries is directed fundamentally at protection. It should provide protection from infection, from added injury, and from future disability and deformity. This protection is afforded by noninterference with the wound, cleanliness of surrounding areas, the application of sterile protective dressings and immobilization in the position of function.

II The general requirements for proper early definitive care are:

A—Thorough evaluation of the injury.

1—Determination of the time, place, causative agent and mechanism of the injury.

2—Determination of the nature and extent of the first treatment given.

3—Determination of infection status: whether the wound is relatively clean, grossly contaminated or with infection established.

4—General nature of the wound, i e., contusion, abrasion, burn, incised wound, lacerated wound, crushing wound, puncture wound, tooth wound, imbedded foreign body, fracture, compound fracture, amputation or combined injuries.

5—Evaluation of structural damage.

a—Degree and extent of surface injury.

b—Source of major bleeding.

c—Evidence of tendon or muscle damage by testing function *against resistance*.

d—Evidence of nerve injury elicited by testing for motor and *sensory* functions.

e—Bone and joint injury determined by x-ray.

f—Discovery and exact localization by x-ray of suspected opaque foreign bodies.

B—Adequate facilities and equipment.

1—Each hospital or clinic should have at least one surgeon who is thoroughly familiar with the anatomy and physiology of the hand and who is prepared to undertake the early treatment of its major injuries.

2—Such treatment should be rendered under strictly aseptic conditions, preferably in an operating room, with careful adherence to aseptic technic in the matter of scrubbing, draping, masking and the use of gloves.

3—An adequate supply of appropriate instruments.

4—Sufficient assistance to assure good exposure.

5—Good lighting.

6—Provision of a bloodless field by means of pneumatic tourniquet or blood pressure cuff.

7—Complete anesthesia for the patient, preferably by general anesthesia.

C—Application of appropriate treatment.

1—Thorough cleansing of a wide area around the wound with the wound protected. (Entire hand and forearm). Shaving, soap and water scrub.

2—Thorough cleansing of the immediate wound area, preferably with soap and water or a bland detergent. Antiseptics should not be used in or on the wound.

3—Careful inspection of the wound and assurance of adequate exposure, by additional incision if necessary, *closely paralleling natural creases.*

4—Thorough toilet of the wound, removing, under inspection, all foreign matter. Excision, by sharp and careful dissection, of all completely devitalized or grossly soiled tissue in the wound surfaces. It is essential that the greatest care be exercised to spare all tissues that may be viable, particularly skin, tendon, nerve and bone fragments.

5—Assurance of hemostasis by ligation of major injured vessels.

6—Repair of injured nerves by end-to-end union with fine interrupted perineural sutures. The uniting of divided digital nerves is important to future function.

7—Repair of other soft tissue injuries, where appropriate, i. e., in clean wounds of short duration, in well-cleaned contaminated wounds of not over eight hours' duration, never in wounds with established infection.

8—Reduction of fractures and dislocations, and retention in corrected position by traction or splinting in the position of function (position of grasp with wrist in dorsiflexion).

9—Application of protective dressing, fingers separated by gauze and hand immobilized to such extent as may be necessary to permit healing, in the position of function (never in the flat position).

10—Administration of antibiotics and protective antitoxin as indicated.

D—After-treatment.

1—Elevation and rest of the hand.

2—Noninterference with initial dressing for a sufficient time to permit healing, unless evidences of suppuration develop.

3—Restoration of skin coverage of denuded areas at earliest possible time. Partial thickness skin grafting is a simple and valuable means of promoting early healing.

4—Early restoration of function for nonaffected parts of the hand by directed *active* motion to the fullest extent that will not jeopardize healing of repaired structures.

5—Restoration of function in affected parts of the hand by directed *active* motion as early as is consistent with full healing and preservation of the repair of damaged structures.

Subsequent articles will deal with the particular treatment of special types of injuries.

Prepared by the American Society for Surgery of the Hand.

RETIREMENT

The prominence recently accorded to retirement and pension plans in labor union negotiations with industry, accompanied for the most part by simultaneous demands for wage increases, brings us inevitably to a thoughtful consideration of the effect of these things upon the medical profession. For the most part, physicians as self-employers must provide for their own needs out of earnings; it is part of the price of independence.

However, provision for old age and retirement necessarily has to be made over a long period of time. During this time interval what happens to the national economy is of the greatest importance. Anyone who has tried to fill a bathtub with the drain improperly stopped is in a position to know the result, but few apparently apply the knowledge thus gained to a consideration of their future under a nationally unbalanced budget.

Deficit spending is an insidious evil, because it causes a slow and relatively unnoticed shrinkage in dollar value. When government borrows to meet current bills, its most convenient source of cash is commercial banks. The fact that they do not have the money on hand is no trouble at all. The banks merely ask Uncle Sam for some IOU's called bonds, and in return they give him a checking account equal to the figures printed on the bonds. Then Uncle Sam writes checks to pay his bills, and everybody is happy. More dollars are put in circulation, but the supply of goods that can be bought remains the same. So, every dollar in the nation loses value. "Dollars of your savings account, your salary, pension, life insurance, and bonds slowly shrivel. During World War II, such wildcat financing shrunk your dollars in half."[1]

A little thought ahead will certainly raise the question in every doctor's mind: What will my accumulated dollars be worth when it comes time for me to retire, if this shrinkage continues?

Already it has become necessary to increase social security benefits and welfare allowances to keep pace with rising costs and shrinking dollar values, and no attempt has yet been made to reduce the enormous public debt. Medical schools and hospitals are facing huge deficits unless they accept subsidy. Powerful labor union pressure is continuing for increased wages, shorter hours, pensions, welfare schemes, in spite of dwindling foreign markets for paid-for, not give-away goods. What price your savings? How long can we spend more than we make without disaster?

If you plan at some time to retire or even to continue working, give these things some thought. Coming elections afford the opportunity for every citizen-taxpayer to make his views known. Will you condone deficit spending and mounting debt by your silence?

Editorial, *N. Y. St. J. M.*, July 15, 1950.

[1] Release No. 117, Committee for Constitutional Government, April, 1950.

The health officer responsible for tuberculosis control in his area should, as an integral part of his work, develop an understanding and working relationship with the social agencies in his community. Such a relationship would certainly benefit both agencies. The social agency will gain an insight into the specialized medical and public health problems associated with tuberculosis control and the health agency will have an opportunity to see the positive contributions which social workers and social agencies can make toward the effective management of tuberculosis patients. Robt. J. Anderson, Chief. — Div. Tubere. — Pub. Health Rep., Dec. 2, 1949.

The great physicians of all time have understood that medicine is not a study of disease, but a study of man: an individual who is a member of a family and who is part of a community . . . . . The purpose of medicine is to make available to all the people, in the greatest possible degree, the achievements of science as they relate to the promotion of health and to the prevention and treatment of disease. W. G. Smillie, M. D., New England J. Med., January 12, 1950.

Health education and sanatorium treatment are our two greatest weapons in fighting tuberculosis. We must remember that each patient with active tuberculosis presents a medical problem, a social and welfare problem, an economic problem and, let us never forget, a public health problem. R. D. Thompson, M. D., Bull. Nat. Tuber. A., Jan., 1950.

+ Editorials +

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W. EDWIN BIRD, M. D. ——— Editor
822 North American Building

ANDREW M. GEHRET, M.D. ——— Associate Editor
1007 Park Place

M. A. TARUMIANZ, M. D. ——— Assoc. & Managing Editor
Farnhurst, Del.

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FLORIDA'S PEPPER GETS SALTED DOWN

The following excerpts from John L. Lewis' magazine are quite revealing and indicative:

On the medical question, labor had better begin to think, because if resentment of the voters to National medicine in one-third of the States proves as beneficial to reactionary candidates as in the case of Smathers, the problem of repealing the Taft-Hartley Law—so long as both are linked in the Truman program—will be three times as difficult.

Regardless of how lightly President Truman may seek to brush off the Pepper defeat, the fact remains that the over-all resentment against Pepper crystallized as a result of Pepper's all-out support of the Truman program.

Pepper had to bear the brunt of all the fault-finding levelled at the Brannan Plan, foreign aid, give-away money, point four, lowering of tariffs, Government extravagance, "Commie" influence in Government, Kansas City Graft, FEPC, Repeal of the Taft-Hartley Law and, of course, National medicine.

The heretofore unorganized vote, as a result of the Truman Medical Plan, the FEPC, the raging "Commie" publicity and bitterly contested Brannan Plan, was activated into resentful political action as never before — resulting in a record vote.

In 44 years of covering political campaigns in the Nation and in many States, your editor has never witnessed such effective and productive quiet solicitation of votes as demonstrated by Florida doctors, druggists, dentists, hospital staffs, insurance companies and pharmaceutical representatives, aided and abetted by other professional men.

Funds were quietly raised. Golf matches, poker games, bridge parties and every form of contact which could be conceived and arranged were executed by the medical, drug and hospital fraternity to convince the people that epidemics would be the order of the day, plus bankruptcy, in the event the proposed Truman Medical Plan — which Pepper was supporting — was enacted into law.

Generally speaking, the people in the East, North and West look upon Florida as a State of aged pensioners in need of medical attention that they cannot afford. This is only partially true as affecting the aged. But they do not constitute an appreciable percentage of the voters.

Pepper was up against an unreasoning wall of voters on this question and the more he said in support of National medical aid the more votes he lost.

Pinellas County (St. Petersburg) is the capital of Florida's retired pensioners, yet Smathers carried Pinellas County 18,244 to 15,906 for Pepper.

In contrast to the Pinellas County vote, live-wire, horse-an-dog-racing, night-club Dade County (Miami, Smathers' home) — which Smathers said he could carry easily by 30,000 majority — was lost by Smathers, the

vote being; Pepper 66,893, Smathers, 65,886, according to unofficial returns.

The labor vote showed up in Dade County, while the labor forces were soundly routed in Duval County (Jacksonville, Florida's only industrial city) — which was the fountainhead of money distribution to finance the Smathers campaign — the vote being: Smathers 42,412, Pepper 32,822.

Just how much money was spent in Smathers' behalf will never be known, but Florida campaigns in recent years have proven to be an orgy of spending.

Although the State population does not exceed 2,500,000, belated reports and confessions show that a total of more than $550,000 was spent in behalf of the successful Gubernatorial candidate in 1948 — and, in addition, a greater total was spent in behalf of the candidates in opposition.

The Smathers-Pepper campaign was of longer duration than the Gubernatorial contest and the Smathers newspaper and radio advertising far exceeded that of the campaign for governor— all of which might furnish a clue of how the money bags were loosened up in behalf of Smathers. The result would be quite interesting and enlightening if some political writer with a flare for statistics would compute the advertising expenditures of the Smathers-Pepper campaigns.

As an aftermath of the campaign, leaders of the CIO and the AFL are busy villifying each other in a name-calling contest in which each side charges the other with failure to properly support Pepper. The railroaders seem to be taking the result with complaceney.

After all is said and done, a simple analysis proves that the Truman program — plus past association with "Commie" leaders — defeated Pepper.

Editorial, *United Mine Workers Journal*, May 15, 1950.

We are extremely pleased to note that such an experienced political observer as the editor of the *U. M. W. Journal* credits the physicians and other professional interests with a major part in this campaign. What Florida can do, Delaware can do, especially now that Mr. Truman has taken another walloping whack in the 249-17 defeat of his Reorganization Plan No. 27, to sneak medicine into his three-ring circus, with Oscar Ewing as the ringmaster. When will he begin to catch on?

SPEAK NOW . . .

Or else hereafter, forever, hold your peace.

In every human undertaking, there comes a time for action, a time for decision. You can describe it in the language of the marriage service, or—if you prefer—of the poker table, "Put up or shut up."

No matter how you phrase it, the alternative of such a time cannot be denied.

This year is a time of decision that requires positive action on the part of the medical profession. If this action is not forthcoming, doctors cannot reasonably complain of the consequences.

This is a year in which the American people elect Senators and Congressmen to represent them in Washington. Under our system of Government, it's up to every citizen to work for the success of candidates in whose views he believes. Only through *active* effort can we have *good* government.

This responsibility is now squarely before all doctors. If they are to be well represented they must work, and they must start now. Doctors, their families, their friends, all they can influence must be registered. On election day—in primary balloting and in November —it's up to the doctors to help turn out the vote—the vote for their candidates.

There is only one way to preserve American freedom—medical freedom—under our democratic process. That way is the voting way . . . the electioneering way. It's the best way ever devised, but it poses responsibilities.

They are responsibilities no doctor can afford to sidestep.

They are responsibilities that need meeting . . . now . . . today.

HELP WANTED

Several towns in Delaware are still in need of additional physicians, notably Georgetown, Milton, Frederica, Felton and Selbyville. In addition we are advised that Dover needs two more general practitioners and two men who would be willing to devote their work

to obstetrics exclusively, as well as an EENT specialist. Georgetown is reported to have subscribed $70,000 to build a clinic, if the medical personnel were available.

This office will be glad to supply what information it has and to help establish contact between prospective physicians and these communities whenever called upon.

THANK YOU, DOCTOR

The Medical Society of Delaware owes its thanks to Dr. John H. Foulger, Director of the Haskell Laboratory of Industrial Toxicology who represented our Society at the decennial meeting of the United States Pharmacopeeial Convention, which was held in Washington, D. C. on May 9 and 10 of this year. Appointed by President Seull, Dr. Foulger attended every session of the convention, and will submit a brief report of this to the House of Delegates next October. This is the first time that Delaware has been represented at the U.S.P. Convention, so far as we know, in twenty years.

READ THIS, TOO

Doctor, when you peruse the advertising pages in our Journal, remember this: all ads are carefully screened—the items, services, and messages presented are committee-accepted. Our standards are of the highest. The advertisers like our Journal—that's why they selected it for use in their promotional program. They seek your patronage and your response encourages continued use of our publication. In turn, the advertisers' patronage helps us to produce a Journal that is worthwhile. When you send inquiries, tell them that you read their advertisement in the *Delaware State Medical Journal*.

The most important factor in the development of the infant mortality rate is the standard of nutrition of the people and the most important factor in the tuberculosis rate is the standard of overcrowding. S. Leff, Med. Officer, Feb. 4, 1950 — Quoted in Am. J. Pub. Health—April, 1950.

Excerpts From AMA Address

In a hard-hitting inaugural address at San Francisco on June 27th, Dr. Elmer L. Henderson of Louisville, Kentucky, new president of the AMA, charged that "the administrative arm of our government has failed us in this generation."

The fighting doctor from Kentucky flatly accused "little men with a lust for power" in the executive branch of the government of seeking to make America "a Socialist State in the pathetic pattern of the socially and economically-bankrupt nations of Europe."

The Administration in Washington, asserted Dr. Henderson, is "sick with intellectual dishonesty, with avarice, with moral laxity, and with reckless excesses." That condition must be changed "if we are to survive as a strong, free people" — and he called upon all of the American people to share the responsibility and to uphold the nation's ideals of freedom.

"Tonight I call upon every doctor in the United States, no matter how heavy the burdens of his practice may be, to dedicate himself, not only to the protection of the people's physical health, but also to the protection of our American way of life, which is the foundation of our economic health and our political freedom."

"American medicine has become the blazing focal point in a fundamental struggle which may determine whether America remains free, or whether we are to become a Socialist State, under the yoke of a government bureaucracy dominated by selfish, cynical men who believe the American people are no longer competent to care for themselves.

"These men of little faith in the American people propose to place all our people, doctors and patients alike, under a shabby, government-dictated medical system which they call compulsory health insurance. But it is not just socialized medicine which they seek. Their real objective is to gain control over all fields of human endeavor — and to strip the American people of self-determination and self-government.

"There is only one essential difference be-

tween Socialism and Communism. Under State Socialism human liberty and human dignity die a little more slowly, but they die just as surely!"

Then Dr. Henderson, declaring that "American medicine has led the world in medical advances, and has helped to make this the healthiest, strongest Nation on the face of the globe," blasted the critics of medicine with this significant statement:

"It is not American medicine which has failed to measure up to its obligations.

"It is not American business nor American agriculture which has failed — nor the fine, loyal working people of America who have failed.

"It is the administrative arm of our Government in Washington which has failed us in this generation!"

Stressing the fact that many already recognize the dangerous trend toward concentration of power in Washington, Dr. Henderson declared:

"If it were not for the leadership of the American press, in defending our fundamental liberties, American medicine, even now, might be socialized — and under the heel of political dictation.

"The newspapers of America, with few exceptions, have taken a strong stand, not only against socialized medicine, but against all forms of State Socialism in this country, and the doctors of America are proud to take their stand beside the fighting editors of America in the battle to save our freedom and the system of individual initiative which maintains it."

Reviewing the great achievements of American medicine at the half-way mark of the 20th Century — with 19 years added to the life span during the past five decades, with many dreaded diseases conquered, which were leading killers at the turn of the century, and with the maternal death rate in this country now lower than in any other nation — the A.M.A. president commented:

"The story of never-ending medical progress in this country is not just a story of so-called miracle drugs and miracle discoveries. The real miracle of American medical progress is the miracle of America itself — the motivating power of the American spirit, of free men, unshackled, with freedom to think, to create, to cross new frontiers.

"This is the spirit, and these are the very methods, which government-domination of medical practice would destroy."

Declaring that the nation's medical care problems can be resolved "without compulsory payroll taxes and without political pressure," Dr. Henderson pointed out that approximately half the population of the country already has enrolled in voluntary health insurance plans "to take the economic shock out of illness."

"Within the next three years, in the opinion of leading medical economists, 90 million persons will be enrolled in the voluntary prepaid medical plans — and when that number has been reached, the problem will have been largely resolved."

Dr. Henderson concluded his address by thanking the American people for coming to medicine's defense when it was brought under attack — and reported that more than 10,000 national, state and local organizations, with many millions of members, have taken positive action against compulsory health insurance.

The hospital is a key location in which to conduct a case finding program. Many of the related factors of tuberculosis control such as nutrition, economic distress and social maladjustment are receiving constant study and attention, but the basic solution remains one of action in mass case finding, diagnosis and treatment. The Importance of the Hospital as a Tubere. Case Finding Center — Hospital Council of Greater New York and New York Tubere. & Health A.—1950.

One of the most persistently discouraging facts about cancer of the lung, is the long interval of ten months that elapses, on the average, between the patient's first visit to the doctor and the time when the diagnosis is made. Overholt, R. H., and Schmidt, I. C., New England J. Med., Nov., 1949.

OBITUARY

SAMUEL C. RUMFORD, M. D.

Dr. Samuel Canby Rumford, 73, retired physician and member of two of Delaware's best known families, died on June 29, 1950 at his home in Wilmington after a long illness.

One of the most prominent physicians in Wilmington for many years, Dr. Rumford was born July 23, 1876, at 1401 Market Street, long known as the old Canby Home, now the site of the Brown Vocational School. He was a son of Charles Grubb and Elizabeth Canby Rumford.

After attending Friends School here, Dr. Rumford attended the Penn Charter School in Philadelphia, and was graduated from the University of Pennsylvania in 1899, and from the Medical School of the University of Pennsylvania in 1902.

After serving his interneship at St. Agnes Hospital in Philadelphia, Dr. Rumford opened practice at 1403 Market Street, and continued practice in medicine and surgery until he retired about 10 years ago.

For 15 years before retiring Dr. Rumford served as medical director of the Continental Life Insurance Company of Wilmington. He served for many years on the staff of the Delaware Hospital.

Dr. Rumford was a former member of the Medical Society of Delaware and of the New Castle County Medical Society. He was also a member of the Society of the Fine Arts and of the Society of Natural History of Delaware, and was interested in the Society of Friends.

Surviving Dr. Rumford are his wife, Mrs. Beatrix Tyson Rumford; a son, Lewis Rumford II of Baltimore; a brother, Lewis Rumford of Wilmington, and four grandchildren, Beatrix Tyson Rumford, Ellen Ellsworth Rumford, Elizabeth Clymer Rumford, and Lewis Rumford III all of Baltimore.

The funeral took place from the home on July 1st, with interment private.

BOOK REVIEWS

A Textbook of Surgery by American Authors. Edited by Frederick Christopher, M. D., of Surgery, Northwestern University Medical School. Pp. 1550, with 1465 illustrations of 742 figures. Cloth price, $13.00. Philadelphia: W. B. Saunders Company, 1949.

We welcome the new fifth edition of Christopher's well known and favorably received book. This edition contains a complete revision of the previous text, written by 198 American authorities with Dr. Christopher as editor and co-ordinator. The book represents practically all phases of American surgery, including orthopedics, urology and gynecology.

This is one of the best one-volume texts in this country, and we recommend it highly.

Harofe Haivri

The appearance of Volume 1, 1950 of the *Hebrew Medical Journal* (Harofe Haivri), inaugurates the 23rd year of its publication under the editorship of Moses Einhorn, M.D.

Written in Hebrew, with English summaries, the *Journal* contributes to the development of Hebrew medical literature, and the newly established Hebrew University-Hadassah Medical School.

The current number contains a symposium on various phases of disease and health in Israel. Among the articles of interest are "Orthopedic Problems in Israel" by I. Pulvermacher, M.D.; "Fighting Deafness in Israel" by Ahron Schwarzbart, M.D.; and "Kupat Holim—The Labor Health Service in Israel" by Moshe Rabinowitz, director of Kupat Holim in Tel Aviv.

In the section on Bible and Medicine, Dr. C. Genazzani presents a unique essay on "Pathological Symptoms Caused by the Famine During the Siege of Jerusalem by Nebuchadnezzar, King of Babylon." In the section on Old Hebrew Medical Manuscripts, Dr. Zussmann Muntner of Jerusalem presents a historical article on "Ascites—A 10th Century Manuscript" by Yizchak Ben Shlomo Ha-Yisraeli. Under the heading of Personalia are presented biographical sketches on the life and works of Professor Hermann Strauss and of Dr. Howard Lilienthal, noted American surgeon.

For more information write to the *Hebrew Medical Journal*, 983 Park Avenue, New York 28, N. Y.

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